AD-P004081

COMPONENT PART NOTICE

THIS PAPER IS A COMPONENT PART OF THE FOLLOWING COMPILATION REPORT:

(TITLE): Proceedings of the AMEDD Psychology Symposium Held at Washington, DC on 27-31 October 1980.

(SOURCE): Academy of Health Sciences (Army) Fort Sam Houston, TX

TO ORDER THE COMPLETE COMPILATION REPORT USE AD-A147 332.

THE COMPONENT PART IS PROVIDED HERE TO ALLOW USERS ACCESS TO INDIVIDUALLY AUTHORED SECTIONS OF PROCEEDINGS, ANNALS, SYMPOSIA, ETC. HOWEVER, THE COMPONENT SHOULD BE CONSIDERED WITHIN THE CONTEXT OF THE OVERALL COMPILATION REPORT AND NOT AS A STAND-ALONE TECHNICAL REPORT.

THE FOLLOWING COMPONENT PART NUMBERS COMPRISE THE COMPILATION REPORT:

AD-P004 081

Proceedings of the AMEDD Psychology Symposium
27-31 October 1980, Walter Reed Army Medical Center

MEETING THE CHEMICAL THREAT
PSYCHIATRIC CASUALTIES IN A CHEMICAL ENVIRONMENT

James A. Romano, Jr.
Behavioral Toxicology Branch
US Army BioMedical Laboratory
Aberdeen Proving Ground, MD 21010

ABSTRACT

If the modern battlefield includes the use of chemical weapons, many new difficulties will arise in the provision of health care, particularly mental health care. The chemical threat may be viewed from both a historical and modern point of view. The status of antidote research is of great concern. Finally the peculiar psychiatric problems which might be foreseen in a chemical environment will be presented. These problems arise from stress reactions, high or low level exposures to anticholinesterases, and high or low level exposures to anticholinergic (antidote) compounds.

INTRODUCTION

The expenditures for protective chemical gear for the US Army has increased 300% from 1969 to the current fiscal year even when measured in constant dollars (Meselson and Robinson, 1980). Why has this happened? What is the perceived threat? What is the impact on the individual soldier? What about problems in the delivery of health care?

In order to properly develop the background of the threat a brief examination of past usage of chemicals in war would be helpful. Joy (1979) defines chemical warfare to include any lethal or damaging effects to men and equipment produced by chemicals, flames, smokes, or obscurants. Its use on the battlefield dates from the Peloponnesian War, in the form of a bellows used to blow smoke from burning pitch across embattlements. The medical community has always considered the treatment of "contaminated" wounds as one of its responsibilities, considering the earliest bullet wounds to be contaminated or infectious, and treating them with cautery. As long as chemicals have been used, though, their effectiveness has been debated.

For example, during the Crimean War a British military man with the unlikely name of Thomas Playfair advocated the use of cyanide bombs. In his words, they would "lessen the suffering of combatants." His advice was ignored and the British suffered the usual 50% casualties in storming fixed fortifications (Joy, 1979).

In the late 19th century, with the development of chemical fertilizers and aniline dyes the chemical industry, especially in Germany, was ready to mass produce chemicals and was moving rapidly to the use of phosphorus in producing more toxic pesticides. In fact, so rapid was this development that the possibilities of modern chemical warfare were foreseen by 1899. An international conference was convened at the Hague and Alfred Thayer Mahan, the naval historian, represented the U.S. Nothing was resolved at that conference.

Thus WWI began with no substantive agreements on the control and use of CW in force. By 1915 there resulted a stalemate between German and Allied forces on a wide front. The time for chemicals had come. At Ypres on 22 April 1915 (at approximately 4:15 PM) "a strange cloud of greenish tint arose from the German lines." The cloud turned out to be a highly toxic chlorine gas and resulted in a long break in the Allied lines. The Germans did not follow up this breakthrough, therefore it is of little military significance. The Allies readily responded with effective defensive measures and a few months later the British introduced phosgene. At that time the main effect of "gas" was thought to be as a threat to the morale of troops in the trenches and the populace back home (Prentiss, 1937).

The use of chemicals quickly escalated. On 12 July 1917 the Germans introduced mustard to the battlefield and quickly produced 20,000 casualties. These casualties primarily showed eye injuries, they were virtually blinded. This agent was persistent, not noticeable, and was both a lung and skin irritant. It required a fully protected soldier. For the first time the question was asked "How effectively will the soldier function when burdened with so much protective gear?"

Other concerns developed. Great burdens were placed on the Army's ability to provide medical care. Although only 2% of "gas" injuries were fatal during WWI, medical support was taxed in two ways. For example, in one case where 281 soldiers were admitted to a field hospital (third echelon of medical care), only 90 of the cases were true gas casualties. The rest were made up of malingerers, host misdiagnoses, and/or "gas mania" in green troops (Joy, 1979). Additionally, not only is there a mass casualty situation but also the requirements for medical care were extensive. Table 1 indicated the duration of hospitalization for WWI "gas" cases.

- - - - - - - - - - - - - - - - - - - -

Insert Table 1 about here

- - - - - - - - - - - - - - - - - - - -

Since 31% of "other" injuries were fatal during WWI we see that CW: (a) doesn't kill many troops, (b) places a great burden on the supply system, and (c) ties up medical personnel.

It is important also to note that the WWI experience was particularly devastating to the Warsaw Pact forces--e.g., the Russian Army experienced the greatest number of chemical casualties (475,000), the greatest number of deaths due to chemical agents (56,000), and the highest proportion of fatalities to injuries. Thus in postulating a European scenario which requires that we maximize fire and maneuver, cover and concealed arms, and continuous land warfare, the Warsaw Pact is thought to envision CW as having four useful effects on a European ground war. CW will: (a) generate mass casualties, (b) stop NATO operations, (c) force NATO troops into a debilitating posture, and (d) deny use of key terrain, esp. highways and airfields (Welch, 1979).

A survey of current literature reveals Warsaw Pact superiority in capacity to use or defend against CW weapons (Hoeber and Douglas, 1979; Meselson and Robinson, 1980; Time, 1980) and also indicates the additional effectiveness these weapons can introduce into the modern battlefield.

- - - - - - - - - - - - - - - - - - -

Insert Table 2 about here

- - - - - - - - - - - - - - - - - - -

Table 2 indicates the additional effectives nerve agent (NA) shells present over conventional artillery shells on the modern battlefield.

Three actions are recommended vis-a-vis the threat of Warsaw Pact chemical superiority: (1) detailed analysis and evaluation of total Warsaw Pact CW offensive capabilities and employment strategies, (2) test, analysis, and evaluation efforts should be undertaken to understand the impact of different dose level exposures on the operation and effectiveness of military missions, and (3) we should rethink the problem of deterrence and response to the use of CW in a chemical conflict.

Meselson et al (1980) advise that high levels of chemical defense raise the scale of CW preparation needed to constitute a major military threat, thus enhancing the effectiveness of verification measures in disarmament treaties.

What is the specific nature of the threat we are required to defend against? Table 3 lists the categories of CW agents which are purported threats and known to be in the arsenal of the Warsaw Pact. These agents are characterized in terms of class of action and persistence. Persistent agents are those which remain in the environment for long periods of time and continue to present a hazard either in the form of a vapor or a liquid. The soldier is exposed to these agents in a number of ways--inhalation, cutaneous contact, or ingestion. The mode of exposure interacts with the amount of agent to determine not only the severity of the signs but also the order in which they appear. Table 4 summarizes the signs and symptoms of NA poisoning. However, symptoms of poisoning may be insidious, as is the case with mustard. Toxic concentrations of this compound are minute, its persistence is legendary, and its symptoms take four or more hours to appear.

Insert Tables 3 and 4 about here

The nerve agents represent a new and more serious threat than CW agents employed in past conflicts. Although there are specific treatment regiments, treatment of NA casualties is likely to be performed in a mass casualty situation. Table 5 depicts the AMEDD casualty treatment and evacuation system. Since all areas up to 150 km are within range of CW delivery systems and CSH and Evacuation Hospitals are tied to airfields, these medical facilities are likely targets for CW attach.

Insert Table 5 about here

Specific treatments are available, e.g., in the case of nerve or incapacitating agents. However they are not antidotes. In the case of NA poisoning atropine blocks and decreases excessive Ach in the body, thus blocking many signs and symptoms; however, it does not reactivate inhibited acetylcholinesterase (AchE). A similar statement may be made about the use of physostigmine to treat BZ intoxication. The progress of antidote research has been held up by failure to describe the fundamental action of, say, GD. Such questions as, "Where does GD go in the body after it is detoxified?" or, "Are all the byproducts of degradtion of GD innocuous?" remain to be answered.

At one point in their paper Meselson et al (1980) argued that even at the current levels of prophylaxis and therapy it is doubtful that current antidotes would significantly reduce casualties "in the sense of soldiers put out of action". They do concede that these measures will save lives and bolster morale.

Let us examine the meaning of this statement--What are the types of casualty we can expect to see in a chemical environment? Table 6 lists the many types of casualty to be found, the stress casualties will be discussed in a later talk.

Insert Table 6 about here

PSYCHIATRIC CASUALTIES

If you'll bear with me, I'd like to make a transformation of the above table to a graph which is limited to psychiatric casualties in two dimensions--those adverse drug reactions produced by NA, those produced by antidotes. A second dimension will be introduced, that of dose level (simply dichotomized into high or low doses). Figure 1 represents the classification scheme.

- -

Insert Figure 1 about here

- -

Are there separate clinical entities which can be derived from this matrix? Let's take a look at the relevant literature. Using atropine as the current antidote and as a representative of the family of anticholinergics, let us examine the effects of atropine use.

Atropine may be used prematurely by the soldier for a number of reasons: (1) he may decide to take it prophylactically, (2) he may overreact and use all three 2 mg atropine injectors carried in his mask case, or (3) it is possible he may use it as a substance of abuse.

Historically atropine was not considered to have abuse potential; however, Shervette, Schyldlower, Lampe, and Fearnow (1979) have reported otherwise. Shervette et al reported on 29 adolescents who abused Jimson "loco" weed and found the following: 10 seeds of the plants contained 1 mg of atropine, 90% of Ss showed hallucinations, 70% of Ss were repeated admissions. Some combative behavior was observed. Mydriasis, dry mucous membranes, tachycardia and flushness were commonly present. No serious complications occurred and hospitalization averaged 1.8 days. All Ss recovered fully.

Headley (1980) summarized data from four papers in which the equivalent of 2 AtroPens or less were administered. He reported such central nervous signs as headache and dose-related dizziness and lack of coordination. Table 7 is taken from a standard pharmacology text, Goodman and Gilman (1970) and cites atropine effects at different doses. If we take 6 mg atropine to be roughly equivalent to 85 ug/kg then the paper by Ketchum, Sidell, Crowell, Agajanian, and Hayes (1973) is directly related to this question These authors reported that the hallucinations, confusion, and incoherence produced by high doses of anticholinergics would best be classified as simple delirium, rather than as "psychotomimetic" or "psychedelic" syndromes. The term delirium was expressly defined to include defects in grasp, failure in sustained mentation, fear or anxious suspicion, misinterpretations, hallucinosis, and restlessness (Ketchum et al, 1973).

- -

Insert Table 7 about here

- -

The effects of anticholinergics are known to be enhanced by sleep loss (Safer, 1970). Also, after high doses of anticholinergics, Ss showed both changes in EEG and behavioral changes. The onset, duration, and termination of the behavioral effects parallel the appearance, persistence, and disappearance of slow EEG activity. These effects are thought to persist for 12-16 hours or more, or in terms of Medevac, possibly until the CSH.

Low doses of anticholinergics do not seem to present noticeable, long-lasting signs. So, following the recommendations of Hoeber et al (1979), we look next to those behavioral effects produced by the anticholinesterases (NA). I have arbitrarily divided these effects into high/low dose effects, the criterion for such a dichotomy probably being the rate at which red blood cell cholinesterase (RBC) is depleted in S. By this I mean that if the rate of cholinesterase depletion is slow, a behavioral tolerance may develop. If it is rapid behavioral effects may be noticeable.

Whereas there is little evidence of the use of NA (specifically) in modern warfare we do have data on anticholinesterase poisoning arising from its use in agriculture (also accidental exposures of industrial workers and animal data). An article by Hayes, Van Der Westhuizer, and Gelfand (1978) reported that in 105 cases of "severe" exposure to organophosphate (OP) pesticides (compounds similar in action to NA) the prominent symptoms were vomiting, abdominal pain, pin-point pupils, respiratory distress, and other "muscarinic" signs. The authors also reported that mortality can be reduced to less than 15% through rapid diagnosis and treatment. The treatment was atropine and obidoxime or 2-PAMCl.

That surviving patients require extensive medical support is substantiated by Walsh, Molloy, and Shanahan (1979) who reported on a patient who had ingested 23g of Malathion. Whereas atropine therapy (28 mg daily for 6 days) was successful, 2-PAMCl did add to efficacy. A significant component of the therapy was IPPV and a nursing staff which was expert in long-term care of patients on respirators. Severe muscarinic signs continued until the 12th day after ingestion. Again, note the problems for combat medical care.

On the basis of studies like these it was determined that severe intoxication by OPs leads to psychiatric sequelae. Wadia, Sadagopau, Amin, and Sardesai (1974) reported that disturbances in consciousness appeared in 10% of exposed agricultural workers. However, these authors felt that such signs as restlessness, emotional lability, nightmares, and confused speech occurred, not as a result of OP poisoning, but as a result of atropine toxicity (which may occur with as little as 6.0 mg in PO exposed patients).

Holmes and Gaon (1956) summarized data on over 600 accidental exposures to OP pesticides. Some severe exposure cases showed an inability to remember street and phone numbers and were unable to recognize old friends. While they could read accurately they were unable to remember what they had read. In summary these authors described the most noticeable features of personality change as (a) forgetfulness and (b) irritability.

Gershon and Shaw (1961) reported on 16 patients chronically exposed to OP pesticides from 1½ to 10 years. Seven of these Ss were reported to develop depression, all showed lapses of memory and concentration. Five were diagnosed as schizophrenic. The authors also concluded that depressive psychiatric disorders were more common in fruit growing areas where OP pesticides were sprayed.

Sidell (1974) reported on four patients accidentally exposed to Sarin and one to Soman. The two who showed the greatest intoxication (one to each compound) had psychiatric problems lasting for several weeks (including sleep disturbances). Since scopalamine ameliorated the mental condition in one patient to whom it was administered, these sequelae were the direct result of excess cholinergic stimulation. Also, the time course of recovery of pupil's ability to dilate was followed in three Ss, this recovery was much the same as that for plasma ChE: initially rapid, with about 2/3 of activity restored in 2 weeks. However, recovery was not completed for several months.

In cases of mild (or low dose) OP exposure some experimental work has been done to go with the clinical data. Investigating the role of the cholinergic system indepressive illness, Davis, Berger, Hollister, and Barchas (1978) reported that administration of DFP to hypomanic Ss produced depression. It did so also in normal Ss (acute low dose exposure). Atropine partially counteracted this effect. The reversible cholinesterase inhibitor, physostigmine, has also been reported to cause depression in some individuals. This effect was especially profound following administration of physostigmine to intermittent marijuana users.

Clinical studies of chronic low dose exposure have yielded mixed results. For example, Wicker, Williams, Bradley, and Guthrie (1979) monitored RBC and plasma ChE in cotton scouts. Although group CheEs were significantly depressed, at times to below 50% of pre-exposure levels, no symptoms of OP poisoning were confirmed. In fact, only a few of the most depressed-ChE Ss complained of not feeling well. The authors suggested that in the dense foliage the primary exposure was to the legs and hips of the scouts, with clothing acting as an occlusive dressing.

The symptoms brought about by low dose exposures are insidious. Richter, Cohen, Luria, Schoenberg, Weisenberg, and Gordon (1980) in a study of Israeli crop dusters reported that early symptoms of parathion poisoning (sweating, nausea, dizziness, and weakness) were indistinguishable from those associated with heat exhaustion. These symptoms, along with blurred vision, occurred before cholinesterase changes when agricultural pilots and ground crews were exposed to parathion levels greater than .005 mg/kg daily.

There is a lack of agreement as to how persistent the psychological changes will be following OP exposure. Grob, Harvey, Langworthy, and Lilienthal (1947) administered DFP daily to volunteers and found symptomatology of insomnia, excessive dreaming, emotional lability, increased libido, paresthesis, visual hallucinations and tremor, along with EEG changes. This was the first such report about EEG. However, such symptomatology usually disappeared shortly after cessation of exposure and EEG returns to normal in 2 weeks.

A group at University Hospitals, Iowa City, Iowa has published extensively on chronic OP exposures. Using a battery of neuropsychological tests including RT, visual memory, language, and paper-and-pencil measures of anxiety and depression, these investigators have generally reported no changes in neuropsychological performance due to OP exposure (Rodnitzky, Levin, and Mick, 1975; Rodnitzky, Levin, and Morgan, 1978; Levin, Rodnitzky, and Mick, 1976). This group has postulated a relative resistance of higher NS functions to mild OP exposure, although Levin et al (1976) did report that commercial pesticide sprayers showed elevated levels of anxiety when compared to matched control groups. They saw no evidence of depressive illness.

When we look at earlier studies of occupational exposures (with less well-protected workers or more toxic agents), a different picture emerges. For example, whereas Barnes (1961), Bildstrup (1961), Bowers, Goodman and Sim (1964) and Stoller, Krupinski, Christophers, and Blanks (1965) questioned the findings of deficits on methodological grounds, they all agreed that subtle behavioral changes, such as impaired memory and concentration, appeared with regularity in exposed men. Tabershaw and Cooper (1966) reported histories of memory difficulty, depression and emotional stability lasting up to six months following low level chronic exposure in 38% of cases studied.

What is the true picture? If we take the EEG research as a focal point I think the data are fairly clear.

Metcalf and Holmes (1969) suggested that OP exposures might lead to chronic EEG changes. They reported that workers with past histories of both OP and chlorinated hydrocarbon exposures, but with no recent exposures had abnormal EEG records and showed "disturbed" memory and attentive processes.

Duffy, Burchfiel, Bartels, Gaon, and Sim (1978) reported even more persistent aftereffects. In their investigations they first reported that in monkeys a single symptomatic exposure or series of subclinical exposures to Sarin produced EEG changes lasting up to one year. In humans, Duffy et al (1978) reported that workers with histories of exposure to Sarin had waking and sleeping EEGs different from those of workers with no exposure history. These differences were still noted one year after the last prior exposure.

Finally, in an unpublished doctoral dissertation McKee (1970) administered the WAIS to workers known to have been exposed to OP (in this case, Sarin) and found a "general withdrawal of interest, a general lessening of intellectual efficiency, and a tendency toward increased carefulness similar to that detected by Bowers et al (1964). These results suggested that chronic OP exposures contributes to an increase in compensatory maneuvers and to decrease in verbal and interpersonal responsiveness.

In summary, modern chemical warfare presents serious medical problems, with the possible appearance of three additional psychiatric syndromes adding to the other (historical) burdens placed on medical care. These syndromes are atropine-induced "delirium," agent produced depressive psychiatric disorders, or, in the case of chronic low dose exposures, a generalized memory and intellectual slowdown with anxiety.

REFERENCES

Barnes, J.M. Mode of action of some toxic substances, with special reference to the effects of prolonged exposure. British Medical Journal, 1961, 5260, 1097-1104.

Bildstrup, P.L. Letters to the editor--psychiatric sequelae of chronic exposure to organophosphorous insecticides. Lancet, 1961, 2, 103.

Bowers, M.B. Goodman, E., & Sim, Van M. Some behavioral changes in man following anticholinesterase administration. Journal of Nervous and Mental Diseases, 1964, 138, 383-389.

Davis , K.L., Berger, P., Hollister, L., & Barchas, J. Minireview: Cholinergic involvement in mental disorders, Life Sciences, 1978, 22 (21), 1865-1871.

Duffy, F.H., Burchfiel, J.L., Bartels, P.H., Gaon, M., & Sim, V.M. Longterm effects of an organophosphate upon the human encephalogram. Toxicology and Applied Pharmacology, 1979, 47, 161-176.

Duffy, F.H., & Burchfiel, J.L. Long term effects of the organophosphate Sarin on EEGs in monkeys and humans. Neurotoxicology, 1980, 1(3), 667-690.

Gershon, S., & Shaw, F. Psychiatric sequelae of chronic exposure to organophosphorus insecticide. Lancet, 1961, 1, 1371-1374.

Goodman, L.W., & Gilman, A. The Pharmacological Basis of Thereapeutics (4th ed.). New York: The MacMillan Co, 1970.

Grob, D., Harvey, A.M., Langworth, O.R., & Lilienthal, J.L. The administration of DFP to man: Effect on the central nervous system with special reference to the electrical activity of the brain. Bulletin of the Johns Hopkins Hospital, 1947, 81, 257-266.

Hayes, M.M., van der Westhuizer, N.G., & Gelfand, M. Organophosphate poisoning in Rhodesig: A study of the clinical features and management of 105 patients. South Africa Medical Journal, 1978, 54, 230-234.

Headley, D.B. A review of the effects of atropine sulfate and pralidoxime chloride on visual, performance, subjective, and cognitive variables in man. Manuscript submitted for publication, 1980.

Hoeber, A., & Douglas, J. The neglected threat of chemical warfare. International Security, 1979, Summer, 3(1), 55-84.

Holmes, J.H., & Gaon, M. Observations on acute and multiple exposure to anticholinesterase agents. Transactions of the American Clinical and Climatological Association, 1956, 68, 68-101.

Joy, R. History of chemical agents in warfare. Paper presented at the meeting of the Treatment of Chemical Casualties Course, US Army BioMedical Laboratory, APG, Md., May 1979.

Ketchum, J., Sidell, F.R., Crowell, E., Agajanian, G., & Hayes, A. Atropine, scopalamine, and Ditran: Comparative pharmacology and antagonists in man. Psychopharmacologia, 1973, 28, 121-145.

Levin, H.S., Rodnitzky, R.L., & Mick, D.L. Anxiety associated with exposure to organophosphate compounds. Archives of General Psychiatry, 1976, 33, 225-228.

Longo, V.G. Behavioral and electroencephalographic effects of atropine and related compounds. Pharmacological Reviews, 1966, 18(2), 965-996.

McKee, J. Intellectual and behavioral correlates of chronic exposure to toxic chemicals. Unpublished doctoral dissertation, University of Denver, 1970.

Meselson, M., & Robinson, J.P. Chemical warfare and chemical disarmament. Scientific American, April 1980, 242(4, pp. 38-47.

Metcalfe, D.R., & Holmes, J.H. EEG, psychological, and neurological alterations in humans with organophosphate exposure. Annals of New York Academy of Sciences, 1969 160 357-365.

Ostfeld, A.M., Machne, X., & Unna, K.R. The effect of atropine on the electroencephalogram and behavior in man. Journal of Pharmacology and Experimental Therapeutics, 1960, 128, 265-272.

Prentiss, A.M. Chemicals in war. New York: McGraw Hill, 1937.

Prentiss, A.M. Poisoning the Battlefield. Time, March 10, 1980, p. 28.

Richter, E.D., Cohen, B., Luria, M., Schoenberg, J., Weisenberg, E., & Gordon, M. Exposure of aerial spray workers to parathion. Israeli Journal of Medical Science, 1980, 16(2), 96-100.

Rodnitzky, R.L., Levin, H.S., & Mick, D.L. Occupational exposure to organophosphate pesticides. Archives of Environmental Health, 1975, 30, 98-103.

Rodnitzky, R.L., Levin, H.S., & Morgan, D.P. Effects of ingested paration on neurobehavioral functions. Clinical Toxicology, 1978, 13(3), 347-359.

Safer, D.J. The concomitant effects of mild sleep loss and an anticholinergic drug. Psychopharmacologia, 1970, 17, 425-433.

Shervette, R.E., Schyldlower, M., Lampe, R.M., & Fearnow, R.G. Jimson "loco" weed abuse in adolescents. Pediatrics, 1979, 63(4), 520-523.

Sidell, F.R. Soman and Sarin: Clinical manifestations and treatment of accidental poisoning by organophosphates. Clinical Toxicology, 1974, 7(1), 1-17.

Stroller, A., Krupinski, J., Christophers, A.J., & Blanks, G.K. Organophophorous insecticides and major mental illness. Lancet, 1965, 1, 1387-1388.

Tabershaw, I.R., & Cooper, W.C. Sequelae of acute organic phosphate poisoning. Journal of Occupational Medicine, 1966, 8, 5-10.

Wadia, R.S., Sadagopau, C., Amin, C., & Sardesai, H.V. Neurological manifestations of organophosphorous insecticide poisoning. Journal of Neurology, Neurosurgery, and Psychiatry, 1974 37 841-847.

Walsh, J., Molloy, J., & Shanahan, J. Organophosphorus poisoning--a case report. Journal of the Irish Medical Association, 1979, 72(2), 532-533.

Welch, T. The chemical threat. Paper presented at the meeting of the Treatment of Chemical Casualties Course, US Army BioMedical Laboratory, APG, MD, May 1979.

Wicker, G.W., Williams, W.A, Bradley, J.R., & Guthrie, F.E. Exposure of field workers to organophosphorus insecticide; Cotton, Archieves of Environmental Contamination and Toxicology, 1979, 8, 433-440.

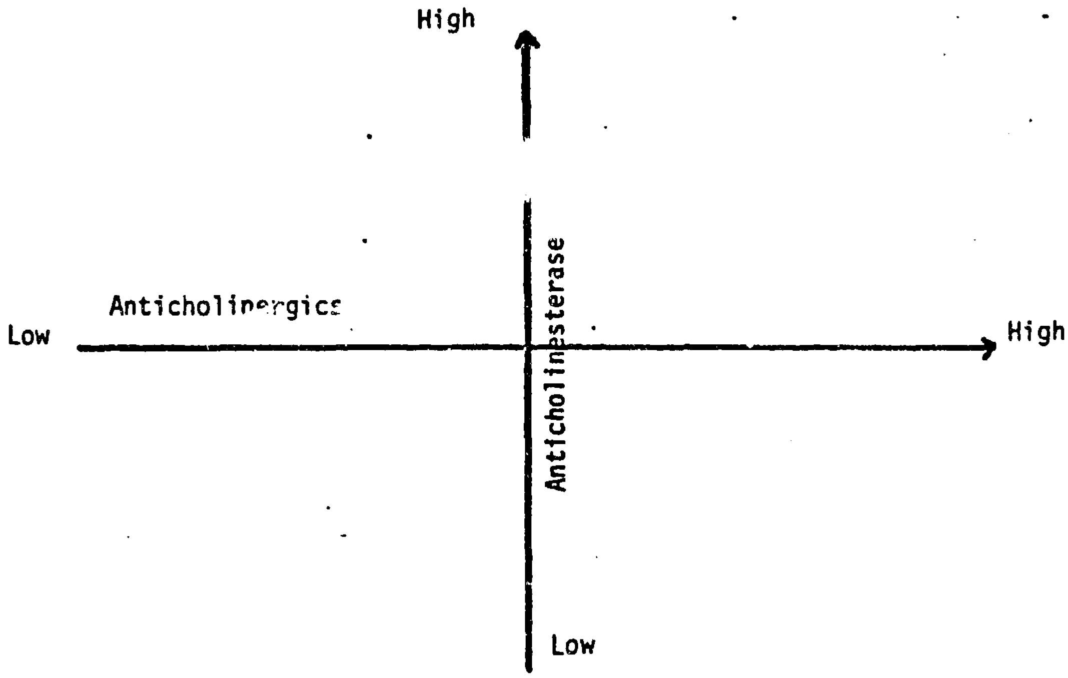


Figure 1. Classification scheme for potential chemical psychiatric casualties.

TABLE 1

Burdens On Medical Care: Hospitalization Due To "Gas" Injuries, WWI

Gas	Casualties	Mean Days Hospitalized
Unknown	33,500	37
Phosgene	6,500	46
Chlorine	1,800	60
Mustard	27,000	46

TABLE 2

Number of Volleys Necessary to Produce 30% Casualties on a Platoon-sized Target at a Distance of 10 km (cool, dry day)

	Nonchemical Shells		Nerve-Gas Shell (GB)			
	Fragmentation Submunition Shell	Airburst High Explosive Shell	Target Personnel Unprotected	Target Personnel Carrying But Not Wearing Gas Masks at Start of Attack	Target Personnel Wearing Gas Masks But Not Protective Clothing	Target Personnel Wearing Gas Masks and Protective Clothing
Target Personnel On The Attach	1	4	1	2	74	(Casualty Level Exceeding A Few Percent Would Be Unattainable)
Target Personnel On The Defensive	4	51	1	66	74	

TABLE 3

Catalogue of Putative Chemical Agents

Type	Agent	Persistency
Nerve	GA (TABUN)	NP
	GB (SARIN)	NP
	GD (SOMAN)	NP
	VX	P
Blister	HD (SULPHUR MUSTARD)	P
	HNI, HN7, NN3 (NITROGEN MUSTARDS)	P
Choking (Lung Damaging Agents)	CG (PHOSGENE)	NP
Blood Agents	AC (HYDROCYANIC ACID)	NP
	CK (CYANOGEN CHLORIDE)	NP
Incapacitating Agents	BZ	NP
	LSD	

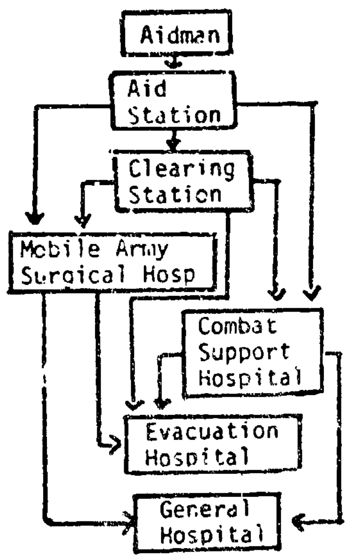
TABLE 4

Signs and Symptoms of Nerve Agent Poisoning

Inhalation (Minutes)
<u>Low Concentration</u>
- Salivation, Rhinorrhea
- Miosis, Dimmed Vision, Accomodation
- Headache
<u>Moderate Concentrations</u>
- Salivation, Rhinorrhea, Trachebroncho Congestion
- Bronchoconstriction
- Miosis, Poor Night Vision
<u>Large Concentrations</u>
- Nausea, Vomiting, Defecation Incontinence
- Altered State of Consciousness
- Convulsions, Fasciculations, Weakness
- Respiratory Impairments

TABLE 5

U.S. Army Medical Department Treatment & Evacuation System



Rearward Evacuation Flow	Evacuation Means	Level of Health Service Support
FEBA	Walking	Unit
	Litterbearer	
5-10KM	Ground Ambulance	
30-40KM	Air Ambulance	
60 KM	Ground Ambulance Air Ambulance	Division
80 KM	Ground Ambulance	Corps
	Air Ambulance	
150 KM	USAF Aircraft	
	USAF Aircraft USN Surface Vessel	COMMZ

TABLE 6

Casualty Types in a Chemical Environment

1.	Pure chemical casualty
2.	Pure conventional casualty
3.	Mixed chemical and conventional casualty
4.	Psychological stress casualty
5.	Physiological stress casualty
6.	Self-inflicted wourds (may include atropine use)
7.	Adverse drug (chemical agent) reactions

TABLE 7

Effects of Atropine at Various Doses in Man

Dose	Effects
0.5 mg	Slight cardiac slowing; some dryness of mouth; inhibition of sweating.
1.0 mg	Definite dryness of mouth; thirst; acceleration of heart, sometimes preceded by slowing; mild dilatation of pupil.
2.0 mg	Rapid heat rate; palpitation; marked dryness of mouth; dilated pupils; some blurring of near vision.
5.0 mg	All of above marked; speech disturbed; difficulty in swallowing; regressness and fatigue; headache; dry, hot skin; difficulty in micturition.
10.0 mg and above	Above symptoms more marked; pulse rapid and weak, iris practically obliterated, vision very blurred; skin flushed, hot, dry, and scarlet; ataxia restlessness, hallucinations and delirium; coma.